The relationship between personal exposure to different levels of benzene and urinary biomarkers.

A A Melikian[1], Q S Qu[2], R Shore[2], G Li[3], S. Yin[3], H Li[1], X Jin[2], B Cohen[2], L C Chen[2]. [1]American Health Foundation, Valhalla, New York. [2]New York University, Tuxedo, New York. [3]Chinese Institute of Occupational Medicine, Beijing, China.

About 60% of smoking-related acute myeloid leukemia mortality is attributed to benzene present in tobacco smoke. An international collaborative study was conducted in an occupational cohort in China to verify the validity, specificity, and sensitivity of biomarkers (*e.g.* chromosomal damage, protein adducts, urinary metabolites) of benzene at low exposures and to assess their relationships with personal exposure and genetic damage. The relationships between personal exposures to different levels of benzene with urinary benzene metabolites in 51 unexposed and 130 exposed workers were investigated. The metabolites monitored in urine were *S*-phenylmercapturic acid (*S*-PMA), *trans, trans*-muconic acid (*t,t*-MA), 1,2,4-trihydroxy-benzene, hydroquinone, catechol (quantified by LC-MS/MS), and phenol (analyzed by GC-MS). *S*-PMA and *t,t*-MA, but specifically the former, correlated well with personal benzene exposure over a broad range of exposure (0.06 to 122 ppm). There was good correlation in the subgroup that had been exposed to <1 ppm benzene (p <0.0001 for *S*-PMA and 0.006 for *t,t*-MA). Furthermore, the levels of *S*-PMA were significantly higher in the subgroup exposed to <0.25 ppm than in unexposed subjects (n=17; p=0.001). The biotransformation of benzene to urinary *S*-PMA ranged from 0.005 to 0.3% and that to urinary *t,t*-MA ranged from 0.6 to ~20 %. Biotransformation of benzene into *S*-PMA and *t,t*-MA decreased remarkably with increasing concentration of benzene. In conclusion *S*-PMA is the best biomarker to benzene exposure. Support: Health Effects Institute and NCI grant P30 CA-17613.